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=> s (androgen receptor) and (codon 741)
        37429 ANDROGEN
        29411 ANDROGENS
        46205 ANDROGEN
              (ANDROGEN OR ANDROGENS)
       794559 RECEPTOR
       732160 RECEPTORS
       951554 RECEPTOR
              (RECEPTOR OR RECEPTORS)
        11973 ANDROGEN RECEPTOR
              (ANDROGEN(W)RECEPTOR)
        42806 CODON
        19224 CODONS
        52021 CODON
              (CODON OR CODONS)
         3679 741
            2 CODON 741
              (CODON(W)741)
L1          1 (ANDROGEN RECEPTOR) AND (CODON 741)

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L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2009 ACS on STN
AN   2003:39630  CAPLUS
DN   139:17235
TI   Novel Mutations of Androgen Receptor: A Possible
     Mechanism of Bicalutamide Withdrawal Syndrome
AU   Hara, Takahito; Miyazaki, Jun-ichi; Araki, Hideo; Yamaoka, Masuo; Kanzaki,
     Naoyuki; Kusaka, Masami; Miyamoto, Masaomi
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CS Pharmaceutical Research Laboratories I, Takeda Chemical Industries, Ltd., Osaka, 532-8686, Japan

SO Cancer Research (2003), 63(1), 149-153
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA English

AB Most prostate cancers (PCs) become resistant to combined androgen blockade therapy with surgical or medical castration and antiandrogens after several years. Some of these refractory PCs regress after discontinuation of antiandrogen administration [antiandrogen withdrawal syndrome (AWS)]. Although the mol. mechanisms of the AWS are not fully understood because of the lack of suitable exptl. models, one hypothesis of the mechanism is mutation of androgen receptor (AR). However, bicalutamide, which has become the most prevalent pure antiandrogen, does not work as an agonist for any mutant AR detected thus far in PC. To elucidate the mechanisms of the AWS, we established and characterized novel LNCaP cell sublines, LNCaP-cxDs, which were generated in vitro by culturing androgen-dependent LNCaP-FGC human PC cells in androgen-depleted medium with bicalutamide to mimic the combined androgen blockade therapy. LNCaP-FGC cells did not grow at first, but they started to grow after 6-13 wk of culture. Bicalutamide stimulated LNCaP-cxD cell growth and increased prostate-specific antigen secretion from LNCaP-cxD cells both in vitro and in vivo. Sequencing of AR transcripts revealed that the AR in LNCaP-cxD cells harbors a novel mutation in codon 741, TGG (tryptophan) to TGT (cysteine; W741C), or in codon 741, TGG to TTG (leucine; W741L), in the ligand-binding domain. Transactivation assays showed that bicalutamide worked as an agonist for both W741C and W741L mutant ARs. Importantly, another antiandrogen, hydroxyflutamide, worked as an antagonist for these mutant ARs. In summary, we demonstrate for the first time that within only 6-13 wk of in vitro exposure to bicalutamide, LNCaP-FGC cells, whose growth had initially been suppressed, came to use bicalutamide as an AR agonist via W741 AR mutation to survive. Our data strongly support the hypothesis that AR mutation is one possible mechanism of the AWS and suggest that flutamide might be effective as a second-line therapy for refractory PC previously treated with bicalutamide.

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